

Patients underwent scintigraphy two, six, and 24 hours after taking 200 MBq of ^{99m}Tc -sucralfate orally with 500 ml of mannitol (the procedure described by Dawson *et al*.¹ Ten of our patients underwent a double contrast barium enema roughly 72 hours after the scan, and the other patient underwent a pancolectomy four days after the scan. Scans were reported on independently by two nuclear physicians, the barium studies by the same consultant radiologist. Each observer drew on a picture of a colon the areas they believed to be abnormal, and a histopathologist did likewise for the colectomy specimen.

The results showed very poor concordance between the two physicians reporting on the same scans by the criteria of Dawson *et al*. There was also poor agreement between the scintiscans and radiology and histology as to the extent of disease. In general, scans suggested more right sided colonic disease than the barium studies, probably owing to labelling of faecal material proximal to the inflamed mucosa.

Two other features led us to abandon this procedure; even after 24 hours scan appearances could change considerably from one minute to the next, and patients, when questioned, said that they found the length of the procedure and the effects of the mannitol more distressing than a barium enema.

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1 Crump BJ, Field S, Rake MO, Kettle AG, Buxton-Thomas MS, Coakley AJ. ^{99m}Tc -Sucralfate imaging in inflammatory bowel disease—poor correlation with radiology. *Nuclear Medicine Communications* 1987;8:273.

2 Dawson DJ, Khan AB, Miller V, Ratcliffe JF, Shreeve DR. Detection of inflammatory bowel disease in adults and children: evaluation of a new isotopic technique. *Br Med J* 1985;291:1227-30.

SIR,—The findings of Mr A George and colleagues (5 September, p 578) disagree with those of Dawson *et al*,¹ whose work also stimulated us to assess sucralfate in imaging colonic disease.

We studied eight patients with right sided colonic carcinoma, a common disease in elderly patients, in whom barium enema examination is often poorly tolerated and consequently may not provide satisfactory imaging of the right colon. A bowel preparation of 500 ml 10% mannitol solution followed by orthograde lavage with saline was used on the day before the study. For imaging we used ^{111}In sucralfate and ^{99m}Tc HIDA as a faecal marker. The vast bulk of imaged activity was in the intraluminal contents held up proximally to the tumours, and after subtraction of Tc labelled faecal material the tumour itself could be visualised in only one patient. The patients all underwent right hemicolectomy the day after the study, and the specimens were imaged on a gammacamera before and after luminal lavage. The tumour usually appeared as a cold spot between levels of high activity, which disappeared after lavage of the intraluminal contents. The ulcerated surface of the tumour did, however, bind the sucralfate to a greater extent than normal mucosa (median ratio 3:2).

While the tumour:mucosal ratios offer some hope for sucralfate in imaging ulcerating colonic lesions, our results show that sucralfate attaches predominantly to faecal contents. George and colleagues postulated that failure of sucralfate to stick to ulcers might be due to not using mannitol in bowel preparation, but our results contradict this view. We were unable to develop a reliable method even though we used a variety of bowel

stimulants and purgatives to promote the passage of the intraluminal contents past the lesion.

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SIR,—We share the disappointment of Mr A George and colleagues, whose scintigraphic imaging with ^{99m}Tc sucralfate failed to reflect the extent or activity of known colonic inflammatory bowel disease.

Using the methods of Dawson *et al*,¹ but omitting the rectal washout, we imaged two patients with extensive ulcerative colitis and 11 with colonic or ileocolonic Crohn's disease, with serial images taken up to 30 hours after ingestion of ^{99m}Tc sucralfate. When compared with the accompanying barium studies and histological findings from six patients who had resections within two to eight weeks of radionuclide imaging scintigraphy underestimated or mislocated active disease in 12 patients; the remaining patient, who had pancolitis, showed diffuse persistent radioactivity throughout the colon. Eight of our patients ingested mannitol and five did not. Results were no better in the mannitol group.

Gastric retention of labelled sucralfate adherent to mucosa has been documented.² This was a major nuisance in seven patients, occurring beyond five hours despite the administration of metoclopramide and exhortations to drink water freely after 30 minutes. The subsequent delayed passage of radioactivity through the gut caused problems with image interpretation.

Disruption of the ^{99m}Tc sucralfate complex, indicated by a faint thyroid and salivary gland image, was generally insufficient to be troublesome. However, the appearance of a gastric image by 23 hours in four patients, including the one with a "true positive" result, was accompanied by a potentially misleading second passage of radioactivity through the gut. The improved stability of sucralfate labelled with ^{99m}Tc DTPA³ prompted its use for a further patient with ileocolonic Crohn's disease. Although thyroid and delayed gastric images were absent, scintigraphy still correlated poorly with barium studies and histology. Bound gastric radioactivity was retained up to six hours.

Thus our experience is similar to that of Mr George and colleagues and suggests that ^{99m}Tc sucralfate scintigraphy is unreliable for assessing inflammatory bowel disease.

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1 Dawson DJ, Khan AN, Miller V, Ratcliffe JF, Shreeve DR. Detection of inflammatory bowel disease in adults and children: evaluation of a new isotopic technique. *Br Med J* 1985;291:1227-30.

2 Carstens AJ, Iturralde M, Fourie PA, Van Wyk A, Pilloy W. Radionuclide studies in upper gastro-intestinal ulceration—are they reliable? *S Afr Med J* 1985;68:867-8.

3 Centi Colella A, Scoparino F. Peptic ulcer imaging by ^{99m}Tc -Sucralfate and possible advantages of ^{99m}Tc -Sucrose octasulfate. *J Nucl Med Allied Sci* 1985;29:192-3.

SIR,—Mr A George and colleagues (5 September, p 578) have had disappointing results using technetium labelled sucralfate to show colonic disease compared with colonoscopy. They question

whether their experience, which contrasts with our own, relates to our use of mannitol, which might favourably affect the colonic pH. Their explanation may well be correct in spite of the fact that they then go on to argue that the alteration of pH produced by the fermentation of mannitol is not likely to be very great. In practice, however, it may be great enough. A further explanation may be that the use of Picolax produces such pronounced intestinal hurry that adhesions to inflamed areas with sucralfate do not occur. Certainly, our paediatric experience using sucralfate to label duodenal ulcers was disappointing, and we had presumed that this was related to transit time.

We should not be surprised that sucralfate is less satisfactory at picking up mucosal lesions than colonoscopy. Colonoscopy is generally accepted to be the investigation of choice for colonic disease.¹ Certainly, colonoscopy and biopsy will produce a higher yield of disease than, for example, double contrast barium enema, which has generally been regarded as the gold standard of radiological examination of the colon.

We have now had considerably more experience with this technique and are re-examining our results. The particular advantages of this technique seem to be that it provides a simple screening test, which is fairly non-invasive and helps to support a clinical diagnosis of inflammatory bowel disease. It also indicates which is likely to be the next major investigation—small bowel enema or colonoscopy. The low radiation dose enables investigations to be repeated when there is doubt about continuing disease—that is, in patients who complain of abdominal pain but whose laboratory results are unhelpful.

We would accept that there are times when uptake of sucralfate by faecal material may cause confusion; this can usually, although not always, be resolved.

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Leucocytosis induced by exercise

SIR,—Dr D A McCarthy and colleagues (12 September, p 636) draw attention yet again to the phenomenon of exercise induced leucocytosis, first described almost a century ago.¹ Pain, extreme emotion, tachycardia, and convulsions all provoke a similar release of white cells into the circulation.² The mechanism, which, as Dr A J Robertson and colleagues point out (3 October, p 856), still requires investigation, probably depends on circulatory changes mediated wholly or in part through catecholamine release. The timing and duration of leucocytosis after exercise seems very variable and has been less fully documented than that after injection of adrenaline, but the biphasic nature of the response is well established^{3,4} and the polymorph leucocytosis that persists for some hours after grand mal seizures has been recognised for over 50 years.^{2,5} Despite the antiquity of these observations, their inclusion in standard textbooks on haematology,^{6,7} and their relevance in many clinical settings, physiological leucocytosis still seems to cause surprise, even consternation, as the following case illustrates.

A 22 year old insulin dependent diabetic in the 35th week of her first pregnancy was admitted to hospital because of hypoglycaemic episodes associated with rapidly falling insulin requirements. At 100 am she was seen to have a grand mal seizure. Hypoglycaemia was confirmed by a fingerprick test, and intramuscular glucagon was administered.

Convulsions stopped (they had lasted for no more than 10 minutes), and subsequent blood glucose measurements were satisfactory. She was nauseated and complained of headache but had no neck stiffness, photophobia, or focal neurological signs. In a blood sample taken at 740 am the same morning the white cell count was $26.2 \times 10^9/l$, 91% neutrophils. A medical senior registrar ordered blood cultures and a viral antibody screen to be performed. At 1100 am her white cell count was $21.4 \times 10^9/l$. Further investigations ordered included culture of nose, throat, rectal, and high vaginal swabs and a catheter specimen of urine. The following morning her white cell count was $12.8 \times 10^9/l$. At no stage did she have fever or any symptoms not attributable to hypoglycaemia. All bacteriological and viral investigations yielded negative results. The patient remained well and at 38 weeks delivered a normal, healthy infant.

Given the combination of circumstances (late pregnancy, hypoglycaemia with concomitant adrenaline release, and a grand mal convulsion), the pronounced leucocytosis and its time course might reasonably have been predicted and the investigations, which were by no means trivial, either to the patient or to the laboratories, avoided.

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District cancer physicians

SIR,—We were surprised at the opinions expressed by Dr L J Donaldson (19 September, p 682) in response to the Association of Cancer Physicians' report recommending the appointment of 63 cancer physicians in district general hospitals in England and Wales.¹

We would be concerned if Dr Donaldson's views were widely held, but we have reason to believe that they are not. Everyone knows that cancer services need improvement; three recent reports²⁻⁴ have emphasised this, and two have stressed the need for more cancer physicians in district general hospitals as well as in university centres, while the Bagshawe report on acute services for cancer⁴ emphasises that "the treatment of cancer patients should be firmly based within the district hospital service." Clearly, the provision of more cancer physicians is only part of the solution. We fully support the view that more radiotherapists are also needed, as are surgeons, gynaecologists, paediatric oncologists, and community physicians with a special interest in cancer.

Dr Donaldson asserts that health authorities are wary of the increased costs that the appointment of cancer physicians would create. This is misinformed, since the appointment of a cancer physician reduces expenditure on cytotoxic drugs and results in their more efficient use (Royal College of Physicians, 1986 (comitia document 86/15)). Furthermore, when a delegation led by Sir Raymond Hoffenberg discussed this issue with the Chief Medical Officer these views were sympathetically received at the Department of Health and Social Security.

Cancer services need improving because patients need better overall care. There is more to care than chemotherapy; flippancy remarks about magic bullets do not advance serious discussion about how the lot of the patient with cancer can be improved. More cancer physicians in the community would help to ameliorate what is currently a totally unacceptable situation.

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SIR,—The proposals of the working party of the Association of Cancer Physicians for a network of cancer physicians, discussed by Dr L J Donaldson, appear to reflect the needs of the patients using our service.

BACUP (British Association of Cancer United Patients) was established two years ago to provide a national cancer information service for patients, their relatives, doctors, and other health professionals.¹ The service is offered by seven trained oncology nurses, who in the past two years have responded to over 30 000 inquiries, largely from patients and their relatives. In a large proportion of our inquiries we encourage and help patients and relatives to go back and speak to their doctors. Our experience shows that patients and their relatives do want in depth information about the disease and its treatment from a reliable and authoritative source. Patients are frustrated by the difficulty in getting such information from their doctors, who are often too busy to spend time dealing with this complex disease. This difficulty is compounded by the lack of cancer specialists, especially in the community. There is an ease in speaking to those who are familiar with the disease, are trained to communicate, and have the experience of the effects of cancer not only on the patients but on their families as well.

We would argue strongly that there is a great need for more cancer physicians, particularly in district general hospitals.

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SIR,—In a well balanced article Dr L J Donaldson has drawn attention to the arguments for a district cancer physician. I would like to make some additional points.

Firstly, radiotherapists and clinical haematologists are specifically examined in their diplomas in the use of cytotoxic drugs. Secondly, I have found my surgical colleagues to be particularly safe in administering cytotoxic drugs, and my chest physician colleagues have lately acquired great skill in the use of cytotoxic drugs with the advent of real advances in treatment, particularly of small cell lung cancer. Thirdly, please remember that we are talking about only a score or so of really usable drugs. Finally, if we do have a district cancer physician I think he will need a diploma which includes a fair chunk of cancer in the examination paper. It would also need to have some statistics, radiation protection, and terminal care as part of its syllabus. Does, I wonder, such a diploma already exist?

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How to take a teaching ward round

SIR,—Mr Alan R Berry (19 September, p 725) indicates the difficulties of teaching small numbers of medical students in hospital wards.

Perhaps it is time for the emphasis of medical student teaching in hospital to change. General practice is now attracting many able doctors and teachers, there are still enough patients in the community to allow teaching in very small groups, and in the community we are not constrained by ward routine.

Judging by the waiting times, there are still plenty of referrals to outpatients. Perhaps medical students should spend their time with these patients in their homes, before either the outpatient appointment or admission to hospital, and be allowed to present the history and examination in the usual way. This would have the additional advantage of providing the students with an understanding of the patients' home environment. It would be expensive in student time, but surely the benefits could be considerable.

I valued my teaching ward rounds as a medical student, but as society progresses so must medical education.

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Emergency phlebography service

SIR,—We wish to comment on the reply to our article (22 August, p 474) from Dr Ian Sykes and colleagues (19 September, p 724). As radiologists we perform those investigations required by the clinicians which are likely to help in their management of the patient. Patients referred for phlebography have a clinically suspected deep vein thrombosis; if the clinician suspects a ruptured Baker's cyst ultrasonography or arthrography is performed. If phlebography shows nothing abnormal it is up to the referring clinicians to determine whether or not further investigation is warranted.

We cannot agree with any of the statements in their last paragraph. Only those patients with a proved deep vein thrombosis will receive anticoagulation so the "unnecessary risk" in patients with the pseudothrombophlebitis syndrome does not occur. The urgent management decision is surely to determine the presence of a deep vein thrombosis, which carries the complications of pulmonary embolism and death.